VISIBLE PULSATION IN RELATION TO BLOOD FLOW AND PRESSURE IN THE PULMONARY ARTERY

BY

MAURICE CAMPBELL

Clinical Assistant to Dr. John Parkinson, National Heart Hospital, 1925–30
From the Cardiac Department, Guy's Hospital and the Institute of Cardiology

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The relief of many cases of cyanotic congenital heart disease by a systemic-pulmonary anastomosis (Blalock and Taussig, 1945) has emphasized the importance of a diminished blood flow to the lungs. The light lung fields and the small pulmonary arteries are most important signs of this. In contrast, the dense lung fields with increased vascular markings and with visible pulsation in the pulmonary arteries and their branches, well known as characteristic of atrial septal defect, suggest an increased blood flow to the lungs.

This division of cases of congenital heart disease into those with a diminished and those with an increased blood flow to the lungs has become so important that in opening a discussion at a joint meeting of the Cardiac and Thoracic Societies in February, 1949, I suggested that the words oligæmic and pleonæmic should be used to describe these lung fields, and should serve, with the presence or absence of cyanosis, as a basis for the classification of congenital heart disease. Fallot's tetralogy and pulmonary stenosis with a closed ventricular septum are the commonest examples of oligæmic lungs: pleonæmic lungs are seen regularly with atrial septal defect, often with ventricular septal defect, and generally but to a lesser extent with patent ductus arteriosus. In cyanotic cases, a similar picture, often in an extreme form with pulsation visible out to the periphery of the lungs, suggests that they too have an increased blood flow; and transposition of the aorta and pulmonary artery—complete or partial—is the most usual anatomical basis for this (Campbell and Suzman, 1950 and 1951). The somewhat similar lung fields that may be seen in mitral stenosis might suggest that they can be due to increased pulmonary arterial pressure rather than to increased pulmonary blood flow. One point raised against the terms oligæmic and pleonæmic was the uncertainty whether these changes indicated increased blood flow or increased pressure.

During the last two years, particular attention has been paid to the lung fields and notes have been made, at the time of screening, about the size of the pulmonary arteries and the degree of visible pulsation. This has been done in several hundreds of cases, but in the majority there have been small pulmonary arteries with a diminished blood flow and no pulsation. The impression formed from the others, i.e. the pleonæmic cases, is that the greatest enlargement of the right and left pulmonary arteries and of their branches, and still more the greatest degrees of pulsation in these vessels are seen with atrial septal defects (and with large ventricular septal defects), and in cyanotic cases with transposition of the aorta and pulmonary artery with septal defects. Similar but slighter changes are found in patent ductus arteriosus: pulsation may be seen easily in the main pulmonary arteries, but much less in the smaller branches and very rarely to the periphery. In mitral stenosis the pulmonary arteries may be wide but there is little or no increase of pulsation.

The most striking examples of this picture, with such obvious pulsation at the hilum that the term hilar dance may justly be used, are seen in a proportion of these cases, mainly with septal defects and with transposition. Though one might expect some additional correlation between a

hilar dance and the presence of a diastolic murmur that might be indicative of pulmonary regurgitation, no close correlation has been found.

These impressions suggest that a large pulmonary blood flow is more significant than a rise of pressure in producing visible pulsation, though both may lead to an increase in size. Before discussing our more detailed findings in support of these general conclusions, a short summary of some earlier and some more recent views has been attempted.

The Pulmonary Arteries in Atrial Septal Defect

Many pathological features of atrial septal defect have been known for a long time, and as early as 1875 Rokitansky emphasized the small aorta and the large pulmonary trunk at necropsy.

Assmann (1929) drew attention to the radiological changes in several types of congenital heart disease. " Enlargement of the pulmonary artery occurs fairly regularly in a condition which to my knowledge has not been mentioned previously in the clinical or radiological literature. The abnormality in question is a large defect of the atrial septum " (p. 89). He mentioned four cases seen by others and added two of his own (one complicated by mitral stenosis); the gross dilatation of the pulmonary artery and its main branches was stressed in both.

Later on he states that in congenital heart disease pulsation can often be seen when the pulmonary artery is dilated (p. 92). " In many cases the hilar shadows are clearly pulsatile. This can be distinguished from transmitted pulsation by the widening and darkening of the shadow." He does not use the term " hilar dance " and does not associate this visible pulsation with any particular type of congenital heart disease. He gave some good illustrations (Plate 2) of the large pulmonary arteries and the increased density of the lungs in both atrial and ventricular septal defects. His Fig. 86 from a patient with morbus cœruleus is very similar to some of our cases of transposition.

Roesler (1934), in a review of atrial septal defect, found that the pulmonary artery and its branches were increased, being on the average half as large again as the aorta. The pulmonary branches at times showed increased pulsation, and were sharply defined and enlarged, but normal lung transparency was present beyond this unless there was congestive failure—a point that we do not accept. He added that these changes might also occur, but less often, with ventricular septal defect and patent ductus, and that this increased pulsation might be due to an increased pulse pressure in the lesser circuit and not always to pulmonary regurgitation.

The Hilar Dance

The first mention of a hilar dance is the description by Pezzi and Silingardi (1925) of a single case. " Dans le champ pulmonaire, on remarque des ombres hilaires énormément dévelopées et sombres surtout à droite, celles de gauche étant en grande partie cachées par la pulmonaire ectasiée. En outre, les ombres hilaires droite présentent des trainées sombres dirigées surtout en bas vers le diaphragme, nettment visibles sur la radiographie, et un phénomène que nous n'avons jamais constanté, c'est-a-dire une véritable danse . . . d'un véritable pouls artérial hilaire." Reading the case notes it seems likely that this patient, with a very large pulmonary artery and a small aorta, had atrial septal defect, though this diagnosis was not made. Laubry, speaking at the meeting where this communication was given thought that these signs must be uncommon with pulmonary regurgitation though he accepted this as the explanation.

Pezzi (1932) wrote further about the " dance of the hilum," which he still thought pathognomonic of pulmonary regurgitation. The cases in which he was interested were those with pulmonary regurgitation secondary to advanced mitral disease, but he added that the same picture was sometimes seen in patent ductus arteriosus.

Recent Views on the Lung Fields in A.S.D. and P.D.A.

There is now widespread agreement about the main features of atrial septal defect (A.S.D.) and patent ductus arteriosus (P.D.A.). The state of the pulmonary arteries and of the lung fields

is recognized as important but somewhat different emphasis is laid on different features. I have tried to summarize the views expressed in some recent writings, omitting the points that are generally agreed, such as the large pulsating aorta in P.D.A. and the small aorta and large right atrium in A.S.D., and emphasizing only comments about the size and degree of pulsation of the pulmonary arteries that are pertinent to the present purpose.

Bedford, Papp, and Parkinson (1941) describing a series of cases of *atrial septal defect* thought the diagnosis rested mainly on the radiological features. The main pulmonary branches are always enlarged. The right branch forms a large dense well-defined and often comma-shaped shadow, contrasting with the clear lung fields, as pulmonary congestion is generally lacking even with congestive failure. The pulmonary artery often pulsates visibly, particularly its right branch (31 of 51 cases), sometimes producing the " hilar dance," independently of the presence of pulmonary regurgitation. Such great pulsation is not always seen, though occasionally a lack of pulsation with increased density implies local thrombosis.

Sussman (1946) thinks that in A.S.D. increased pulsation of the pulmonary arteries and hilar vessels is usual, and that the pulsation is vigorous and of increased amplitude, giving the impression of a hilar dance, presumably due to pulmonary incompetence.

Brown (1950) thinks there is a characteristic X-ray picture for A.S.D. though in children it may take some years to develop: a large rather globular heart, a prominent bulge of the pulmonary arc, and a pulmonary artery dilated almost to aneurysmal proportions, with striking pulsation, especially of the right hilum, and often a hilar dance.

In the description of *patent ductus arteriosus* I have found no general statement that pulsation of the pulmonary arteries is much less than in A.S.D., though I think this is the view of many cardiologists. Roesler (1943) says that the pulmonary arc may be moderately prominent and the vessels of the hilus may be normal or more often enlarged. The pulsation of the main pulmonary artery is increased but expansile pulsation only occurs with pulmonary regurgitation.

Gilchrist (1946) states that enlargement of the pulmonary trunk is usually visible as a semicircular shadow lying between the aortic knuckle and the left ventricular border; in the right oblique this prominence may be more obvious. Other signs are dilatation of right and left pulmonary arteries: " congestion of the lung fields as shown by a diffuse mottling radiating out from the hilum towards the periphery, the ' hilar dance,' a systolic expansion in the arteries of the lung root, the last being uncommon in my experience." Sussman (1946) thinks that the pulmonary artery is dilated in half the cases and pulsation greatly increased, but that a collapsing type of pulse with hyper-active pulsation of the hilum is not usual.

Brown (1950) thinks that if the shunt is large there is much dilatation of the pulmonary artery extending to its branches, with vigorous pulsation of the aorta and pulmonary artery, the latter showing a hilar dance. Kerley (1951) gives a good description of the lung fields in mitral stenosis but gives little about these pleonæmic changes in congenital heart disease and hilar pulsation is said to indicate a high pulmonary pressure.

Taussig's views. Taussig (1947) gives a clearer picture of the relative importance of these radioscopic signs in congenital heart disease. She describes the pulsation in the hilum of the lung, the so-called dancing hilus, under two conditions—when the contraction of the left atrium is reflected back into the pulmonary veins, and when the forward pulsation from the right ventricle becomes visible in the dilated pulmonary arteries. The former is seen with mitral regurgitation and sometimes with severe mitral stenosis: the latter is seen with A.S.D. and with P.D.A., in both of which the main pulmonary branches may be greatly dilated. She states that " In P.D.A. pulsation may be visible at the hilus of the lungs and there may be a hilar dance, but the most conspicuous pulsation occurs with A.S.D. and a true dancing hilus makes this diagnosis most likely."

Later she states that with the Eisenmenger complex and sometimes with a large ventricular septal defect pulsation may be seen at the hilus and in the lung fields, due to the greatly enlarged pulmonary vessels. Taussig thinks that these changes occur when the pulmonary pressure is abnormally high, but elsewhere it is clear she regards them as indicative of an increased pulmonary

blood flow. Even in Taussig's writings there is no sharp distinction made between pulsation at the hilum and a hilar dance. We think that unless the expression is to be reserved for the more conspicuous pulsation there is no object in its use.

Our Present Observations

Compared with the attention that has been paid to the lung fields, little has been written about them, though there is an excellent paper by Lodge (1946). The anatomy of the lung, especially of the hilum, must be considered shortly. Owing to the air they contain, the bronchi may show in a print as a white circle, sometimes enclosed in a ring that represents the walls. The arteries and veins might be expected to show as equally dark shadows, apart from any increase caused by the greater toughness of the arterial wall. This is not so, and a catheter passed into a pulmonary vein appears to lie directly in the lung, while one in an artery is seen surrounded by the arterial contents. The main shadows of the hilum are the pulmonary arteries, as was demonstrated by Assmann (1929) and amply confirmed by Cottentot and de Balzac (1936) and more recently by angiocardiography. The reason is probably because there are four veins instead of two and they divide quickly, so that when they emerge from behind the heart they are already much smaller than the pulmonary arteries. Their shape is more like the spokes of a wheel, and shadows in this position should be looked for, though we think that most of the shadows discussed in this paper are arterial.

We have tried to relate the appearance of the lung fields on radioscopy and on films to the pressure in the pulmonary artery and to the pulmonary blood flow in patients where these data have been obtained by cardiac catheterization. Notes about the appearance of the lung fields were dictated at the time in the X-ray room, almost always before we knew the results of cardiac catheterization, and often several times at intervals up to one or two years.

We have taken the right pulmonary artery* as the best measure of the degree of dilatation of the pulmonary system. The pulmonary trunk may be dilated beyond a stenosis and its apparent size may be modified by the shape of the cardiac shadow. The left pulmonary artery is too often hidden by the heart, and the smaller branches of the right pulmonary artery cannot as a rule be seen with the same precision for an accurate estimate. We measured the diameter of the right pulmonary artery comparing it with the size of the chest, but accuracy was difficult and it seemed better, on the whole, to mark the artery as +, ++, +++, etc., corresponding to the description of slightly, moderately, or greatly enlarged. Even then, consistency of judgement of size was not easy, but many of the original notes have been checked by comparison of the films. Exact consistency as regards visible pulsation is also difficult but the variation at two examinations should not be great.

We have used the same signs for the degree of pulsation and have recorded this as it is seen in the right pulmonary artery, in its main branches in the middle third* of the lung, and in its smaller branches in the peripheral third of the lung. There must be a pulmonary pulse normally, but it is not easily seen even in the main vessels, and a single + indicates something that is abnormal, though (+) indicating a pulse that can just be seen may be within normal limits in the right pulmonary artery. Normally, pulsation can never be seen beyond this, so that any marking in the middle or peripheral thirds is abnormal.

Unless the term hilar dance is restricted to mean something more than pulsation at the hilum, there is no object in its use. We think that it is best defined as a degree of pulsation striking enough

* We have followed *Gray's Anatomy* (1949) in the use of the term "pulmonary trunk" but to save constant repetition of the "descending branch of the right pulmonary artery" we have used the term "right pulmonary artery" to include this descending branch after it has given off the branch or branches to the upper lobe and the branch to the middle lobe but before it becomes greatly reduced in size by dividing into the restrocardiac, anterior basal, axillary basal, and posterior basal arteries (Lodge, 1946). These named branches are the ones we have examined for pulsation in the "middle third" of the lung. The smaller branches in the "peripheral third" are not normally seen as individual vessels.

to be noted in both hila simultaneously, even when one's attention is directed mainly to one hilum, often with an added jerky quality.

Patent Ductus Arteriosus. We have included 10 cases of patent ductus arteriosus, most of them thought to have large shunts as judged by the wide pulse pressure and by the size of the heart, though without much in the way of symptoms. The pulmonary flow averaged 12·3 litres/sq.m./min. and only in two of these was it less than 8 litres/sq.m./min. It is surprising that patients with such large flows as 23 and 16 litres/sq.m./min. should have so few symptoms; it confirms the view that a large left-to-right shunt is often well supported up to the time that heart failure is imminent.

The pulmonary pressure* was never very high, but in five of the patients it was slightly raised, i.e. in three with mean pressures about 45 and in two with pressures of 43/30 and 38/27. In four of these five the pulmonary flows were the most increased. There was close agreement in marking these right pulmonary arteries as ++, which is somewhat less than the estimate of the size made in atrial septal defect, and about the same as that made in mitral stenosis (see Table I). The degree of pulsation was rather more variable, but pulsation was always visible in the right pulmonary artery and, of course, in the pulmonary trunk. Generally it was only marked +, but in two cases as ++. In more than half, pulsation could be seen beyond this out into the middle third, but generally this was marked (+) which means that it could only just be seen. Pulsation was never seen out to the periphery except doubtfully in one case. In none was there anything that could be called a hilar dance.

TABLE I

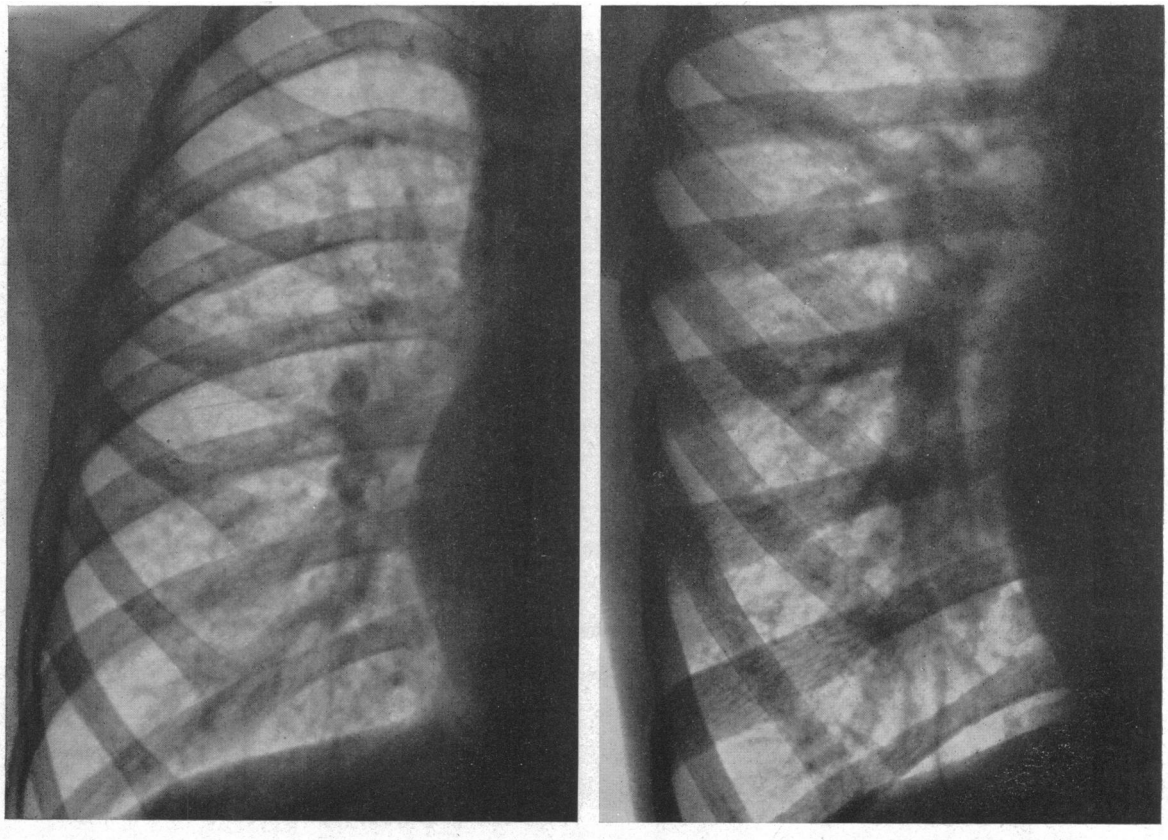
RELATIONSHIP OF SIZE AND DEGREE OF PULSATION TO BLOOD FLOW AND PRESSURE IN THE PULMONARY ARTERIES IN CASES OF PATENT DUCTUS ARTERIOSUS AND ATRIAL SEPTAL DEFECT

Case No.	Age and Sex	Pulm. flow l./sq.m./min.	Pressure (mm. Hg.)	Size of Right P.A.	Pulsation			
					Right P.A.	Middle third	Peripheral third	Hilar dance
			Patent Ductus Arteriosus					
1 (0418)	F7	+++	38/27	++(+)	+	(+)	—	—
2 (P250)	F11	23·0	45	++	+	—	—	—
3 (P255)	F7	16·1	45	++	+	+	—	—
4 (P226)	F5	12·9	43/30	++	++	+	—	—
5 (H279)	F17	11·4	32/15	++	++	+	—	—
6 (O304)	F8	9·6	28/23	+	+	(+)	—	—
7 (O413)	F8	8·6	23/12	++	+	+	(+)	—
8 (O423)	F12	8·5	45	++	+	—	—	—
9 (O381)	F19	6·2	20	++	+	—	—	—
10 (O280)	M27	4·4	18	++	+	+	—	—
			Atrial Septal Defect					
11 (O132)	F10	20·5	42/?	++	++	+	(+)	(+)
12 (O125)	F9	18·0	44/?	+++	+++	++	(+)	+
13 (O305)	F9	12·1	26/7	+++	++	+	—	—
14 (O642)	F48	11·4	33/?	++	++	++	+	+
15 (O112)	M11	11·2	35/?	++	++	+	—	—
16 (H281)	F37	9·1	12	+++	+++	++	+	+
17 (C227)	M27	3·2	72	+++	++	++	+	(+)
			Pulmonary Veins Draining into Right Atrium					
18 (P230)	M8	12·5	30/8	++	++	+	+	—
19 (P256)	F36	9·5	16	++	++	+	+	+
20 (O392)	M19	9·0	50/17	++	++	+	(+)	—

None of these patients had signs of pulmonary regurgitation. All had an arterial oxygen saturation above 94 per cent, except Case 17, where it was 91 per cent.

* Most of the pressures have been measured from a reference level half-way between the skin of the back and the sternum.

Considering that these pulmonary flows cover about the same range as those in atrial septal defect, the smaller increase in the size of the arteries and in the degree of pulsation is striking. We think this is a valuable point in the diagnosis of doubtful cases, with the other signs that are more widely agreed. Two examples of the lung fields, one with an unusually large right pulmonary artery, are shown in Fig. 1. The medium-sized mottling and the visibility of the smaller pulmonary branches are the main features.



A B

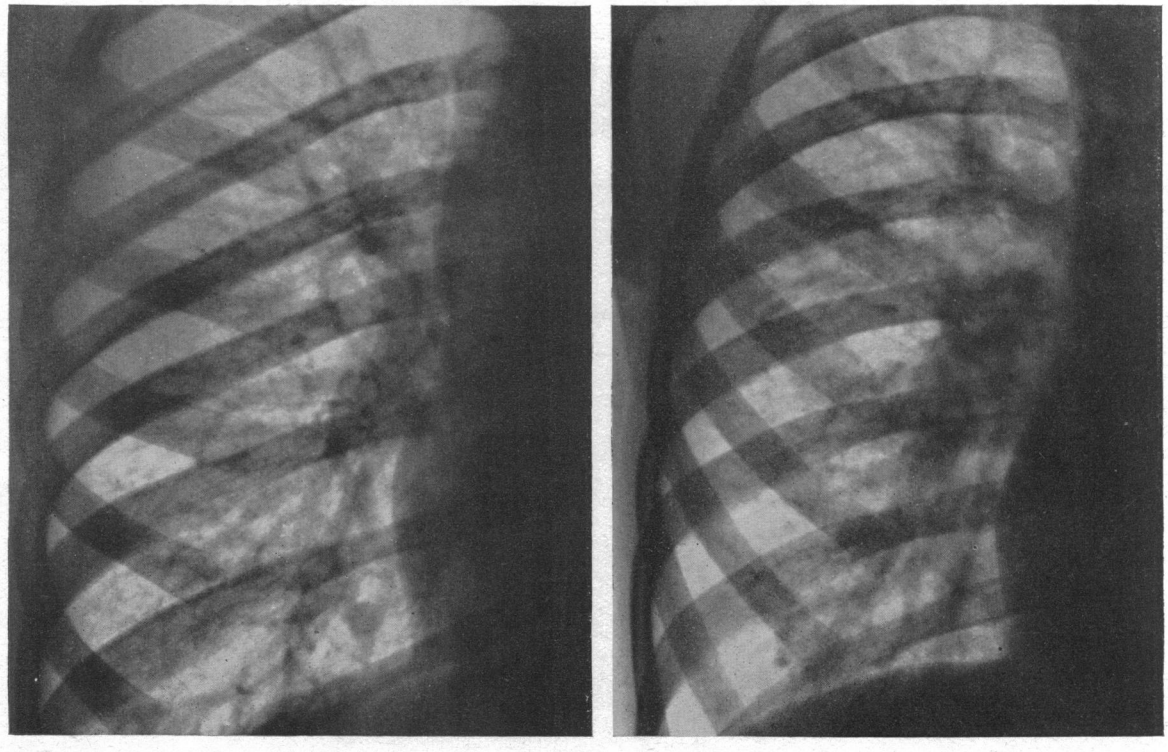
FIG. 1.—The pleonæmic right lung fields from two patients with patent ductus arteriosus.

(A) From a girl, aged 7, with much mottling through the lungs and with a moderate-sized right pulmonary artery. Her heart was not very large, c.t.r. 52 (m.t.d. 10·0/18·5 cm.) in spite of her having a very large flow through the ductus: it could not be calculated accurately owing to the very high arterial oxygen saturation in the pulmonary artery. Case 1.

(B) From a girl, aged 11, with equally mottled lungs and a much larger right pulmonary artery. Her heart, c.t.r. 63 (m.t.d. 12·1/19 cm.), could be seen beating with unusual force through her frail thin chest; nine months after operation the c.t.r. was reduced to 55 (11·3/20·5 cm.). Case 2.

Atrial Septal Defect. The number of cases of A.S.D. where we have used cardiac catheterization is not large, partly because until recently we have not been concerned about their surgical treatment, and partly because in several of the children chosen the septal defects were, in fact, ventricular. Of the 7 cases, 3 were the ordinary adult type with increasing symptoms and the others were children with little disability although they often had moderate, and in one case gross, enlargement of the heart. Three other cases where pulmonary veins drained into the right atrium have been included (see Table I).

The pulmonary flows averaged 11·7 and generally ranged from 9 to 18 litres/sq.m./min.; in most of the children they were even larger than in the adults. The only exception was Case 17



A B

FIG. 2.—The pleonæmic right lung fields from two patients with atrial septal defect. The pulmonary branches can be seen rather better than in Fig. 1A with P.D.A. and there was a greater degree of visible pulsation.
(A) From a girl, aged 10, with few symptoms and moderate enlargement of the heart, c.t.r. 55 (m.t.d. 11·5/21 cm.) Case 11.
(B) From a girl, aged 9, with no symptoms but with a larger heart, c.t.r. 58 (m.t.d. 10·8/18·4 cm.). Case 13.

who has since died with confirmation of the diagnosis at necropsy: probably the rising pressure on the right side of the heart had gradually reduced the original large flow, as it is unlikely that the extensive thrombosis in the pulmonary arteries found at necropsy had been present at the time of his catheterization three years before.

The pulmonary pressures were perhaps a little higher than in many reported cases but they were chosen as fairly extreme examples. They ranged from 26/7 to 44/? and so are hardly outside normal limits. As with P.D.A., the pulmonary flow may be greatly increased with little if any rise of pulmonary pressure.

As a group these patients had slightly larger pulmonary arteries than those with P.D.A. and a much greater degree of pulsation therein than any other group except V.S.D. and transposition. The right pulmonary artery was always marked as at least ++ and more often as +++, and the same applied to the degree of pulsation in these. Pulsation was always easily seen out to the middle third and was equally often marked ++ and +. In more than half the cases pulsation could be seen out in the peripheral third and often easily, and in half there was a striking hilar dance. Two examples of the lung fields are shown in Fig. 2; coarse mottling and large, unusually visible, pulmonary branches are the main features.

These more striking signs on radioscopy can not be due to the average pulmonary flow in A.S.D. being larger than in P.D.A., but must depend on other hæmodynamic factors (see discussion). The only cases with any comparable picture are those with ventricular septal defect or with transposition of the aorta and pulmonary artery, and in the latter the lung fields sometimes show an even greater increase of density.

The three cases with some pulmonary veins draining into the right atrium showed a similar picture on radioscopy and in the great increase of pulmonary flow without much rise of pressure. There were three other cases where the proved presence of pulmonary stenosis did not prevent the pulmonary flows from being increased by left to right shunts to from 5 to 11 litres/sq.m./min. None of the three showed any unusual pulsation, presumably because of the pulmonary stenosis, although the clinical features and the mixed findings in the lung fields had made it difficult to decide whether the lungs were oligæmic.

Ventricular Septal Defect. The classical picture of the small V.S.D., the maladie de Roger, is well known. The importance and frequency of larger defects has recently been emphasized by Taussig (1947), Selzer (1949), Marquis (1950), and Wood (1950*a* and *b*). We agree that they are much more common than has generally been thought and they are the only type of V.S.D. we have met with frequently on catheterization.

We have included in this group 10 cases where the patient was clinically acyanotic and there was a left to right shunt through the V.S.D. In Case 29 this shunt was small but in all the others it was large. The border line between V.S.D. and Eisenmenger's complex is not easy, and some may think that Cases 26 and 27 with lower O_2 saturations should be included as Eisenmenger's complex, but in each the left to right shunt through the V.S.D. was the main feature.

The general findings and the picture of the lung fields in V.S.D. are very similar to those in A.S.D. The pulmonary flows were also within the same range and averaged 12·1 litres/sq.m./min., but there was much more often a rise of pulmonary arterial pressure, sometimes nearly up to the systemic pressure. Only two patients failed to show much rise: in Case 30 the defect was small and the pulmonary flow was not much increased and in Case 25 there were signs of aortic incompetence and we think an ordinary V.S.D. though possibly the aortic sinus had ruptured into the right ventricle (see Table II).

The pressure was raised in all the others but in two patients of three and seven years with large pulmonary flows of 13 and 17 litres/sq.m./min. the pressures were not much above 50/20. In the others, even in one boy aged 5, the pressure was as high as 88/50 or more, and in one girl of 18 as high as 102/61. Generally, a rise of pressure to this level goes with much disability, but this girl was leading an active life and was investigated because of the size of her heart.

The picture on radioscopy, both in the size of the pulmonary arteries and in the degree of pulsation, hardly differs from that described in A.S.D. Striking pulsation in the middle third of the lung was seen just as often, as was pulsation out to the periphery and a hilar dance. Two examples of the lung fields are given in Fig. 4. In (A) the pulmonary branches were unusually dilated, and in (B) there was heavy mottling, some of which may have been due to venous congestion.

Eisenmenger's Complex. In the patients diagnosed clinically as Eisenmenger's complex in the cyanotic stage, it may be difficult to prove the presence of a V.S.D. probably because the rising pressure on the right side has diminished the left to right shunt and allowed a right to left shunt to develop or increase. Corresponding to this, the pulmonary flows were always low normal, or reduced, and in these six patients averaged only 2·5 litres/sq.m./min. (see Table II).

In this group the pulmonary artery was even larger than in A.S.D. and V.S.D. Pulsation was always obvious in the right pulmonary artery and of about the same degree as in A.S.D. and V.S.D. In the middle third it was rather less though always present; it was much less commonly seen out to the periphery and a hilar dance was not seen.

Case 36 is an exception that is difficult to explain: a septal defect was diagnosed with confidence on the shape of the heart and the extreme degree of pulsation visible through the lung fields; there was even a faint hilar dance. Her pulmonary flow was only 2·9 litres/sq.m./min., so that if these signs in the lung fields depend only on the increased pulmonary flow she should not have shown them. She had signs of gross pulmonary regurgitation and this may be a partial explanation.

Primary Pulmonary Hypertension. Four cases have been included in this group. In three there was no reason to suspect congenital heart disease, but in Case 37, there was also a reversed shunt through a P.D.A., confirmed by necropsy (Campbell and Hudson, 1951). The size of the

TABLE II

RELATIONSHIP OF SIZE AND DEGREE OF PULSATION TO BLOOD FLOW AND PRESSURE IN THE PULMONARY ARTERIES IN CASES OF VENTRICULAR SEPTAL DEFECT AND PULMONARY HYPERTENSION

Case No.	Age and Sex	Pulm. flow l./sq.m./min.	Pressure (mm. Hg)	Size of Right P.A.	Pulsation				Arterial O_2 Sat.*
					Right P.A.	Middle third	Peripheral third	Hilar dance	
				Ventricular Septal Defect†					
21 (O634)	F11	25·0	93/40	++	++	+	+	+	98
22 (P233)	M7	17·4	53/20	+(+)	++	+	—	—	98
23 (O142)	M11	17·1	83/35	++	++	++	+	+	97
24 (O163)	M3	13·5	57/27	++(+)	++	++	++	—	98
25 (O545)	F15	11·1	33/8	++	+	+	—	—	98
26 (P252)	M5	10·2	88/50	++	++	++	+	+	89
27 (O467)	F48	9·4	128/58	+(+)	+++	++	+	+	87–99
28 (O606)	F18	8·8	102·61	++(+)	+++	++	+	+	99
29 (O165)	F11	5·5	78	++	++	++	++	+	92
30 (O298)	F8	3·0	24/12	++	++	+	—	—	98
				Eisenmenger's Complex					
31 (P036)	F44	2·2	122/55	+++	+++	++	+	+	79–82
32 (O261)	F24	2·1	95/55	++++	++	+	—	—	79–96
33 (O178)	F37	2·1	155/95	+++	+	(+)	—	—	87–98
34 (O625)	F33	2·0	115/55	++	+	—	—	—	80–88
35 (P092)	F8	3·6	118/?	+++	++	+	—	—	84
36 (O395)	F29	?2·9	58	++++	+++	++	++	(+)	85
				Pure Pulmonary Hypertension					
37 (H104)	F37	?1·8	100	++	++	+	—	—	64–78
38 (V.B.)	M22	1·3	128/83	++(+)	++	+	—	—	90–99
39 (M.L.)	F20	3·6	85/50	++	—	—	—	—	76–98
40 (D.K.)	M25	2·4	102/45	+++	+	(+)	—	—	97

* The second figure indicates the value after breathing oxygen.

† All these, except Case 30, had evidence of a large left to right shunt through a V.S.D. In those grouped as Eisenmenger's complex there was no evidence of a left to right shunt except a small one in Case 31

TABLE III

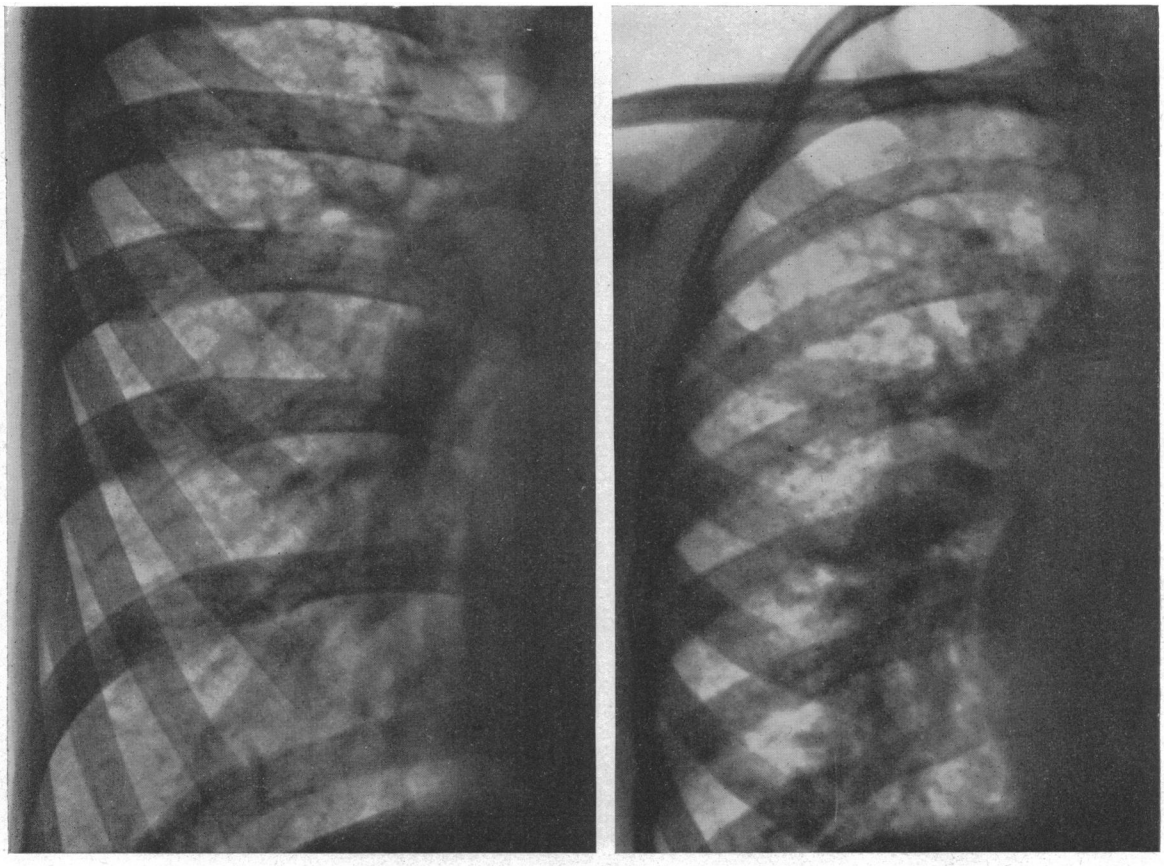
RELATIONSHIP OF SIZE AND DEGREE OF PULSATION TO BLOOD FLOW AND PRESSURE IN THE PULMONARY ARTERIES IN CASES OF TRANSPOSITION*

Case No.	Age and Sex	Pulm. flow l./sq.m./min.	Pressure (mm. Hg.)	Size of Right P.A.	Pulsation			
					Right P.A.	Middle third	Peripheral third	Hilar dance
41 (O052)	M13	+++	(55)†	++++	++	++	++	+
42 (O076)	M16	5·5	88	+++	++	++	++	+
43 (O109)	M14	6·7	94/53	+++	++	+	(+)	—
44 (O175)	M13	?4·1	(85/8)†	+++	++	++	++	+
45 (OA06)	M6	8·3	80/50	++	++	+	—	—
46 (O145)	M7	3·0	(89/16)†	++	+	+	(+)	(+)
47 (P138)	F6	9·4	90/60	+++	++	++	+	—

* Six of these are discussed fully by Campbell and Suzman (1951). † Right ventricle only.

pulmonary artery was less than in cases with about the same pressure diagnosed as Eisenmenger's complex. The degree of pulsation was much less—about the same as in P.D.A.—being present in the right pulmonary artery and sometimes to a lesser degree in the middle third of the lung, but never out to the periphery and never enough to produce a hilar dance (see Table II).

Transposition of Aorta and Pulmonary Artery. There were 7 cyanotic cases where full investiga-



A B

FIG. 3.—The pleonæmic right lung fields from two patients with transposition of the aorta and pulmonary artery.

(A) Large pulmonary arteries and visible branches out to the periphery, with moderate mottling, from a boy, aged 16, where the diagnosis was confirmed by catheterization. Heart moderately enlarged, c.t.r. 55 (m.t.d. 11·0/20 cm.). Case 42.

(B) From a girl, aged 7, showing a larger right pulmonary artery and a greater degree of general mottling. The heart was larger, c.t.r. 60 (m.t.d. 10·4/17·3 cm.). Diagnosis not proved by catheterization. Case P090.

tion supported this diagnosis. All showed a considerable increase of pulmonary arterial pressure which, of course, must be the same as that in the left ventricle, and most of them had some increase in the pulmonary flow, though the figures are less reliable than in simpler cases.

The pulmonary artery was as dilated and there was as much pulsation as in any group. In the right pulmonary artery pulsation was nearly always marked ++; it was always seen in the middle third of the lung and except in one case was visible out to the periphery. In four cases there was an obvious hilar dance (see Table III). The lung fields of two cases are shown in Fig. 3: both show mottling and very large right pulmonary arteries; in one the dilated rather straight pulmonary branches are very striking and in the other, the rounded mottled shadows.

MITRAL STENOSIS

Mitral stenosis is pertinent to the present discussion because there is often a rise of pulmonary arterial pressure with a normal or diminished pulmonary flow. We have included 20 cases for comparison with the pleonæmic lungs of congenital heart disease. There was not much variation in the pulmonary flows, all of which were between 1·5 and 3·2 litres/sq.m./min., except Cases 53

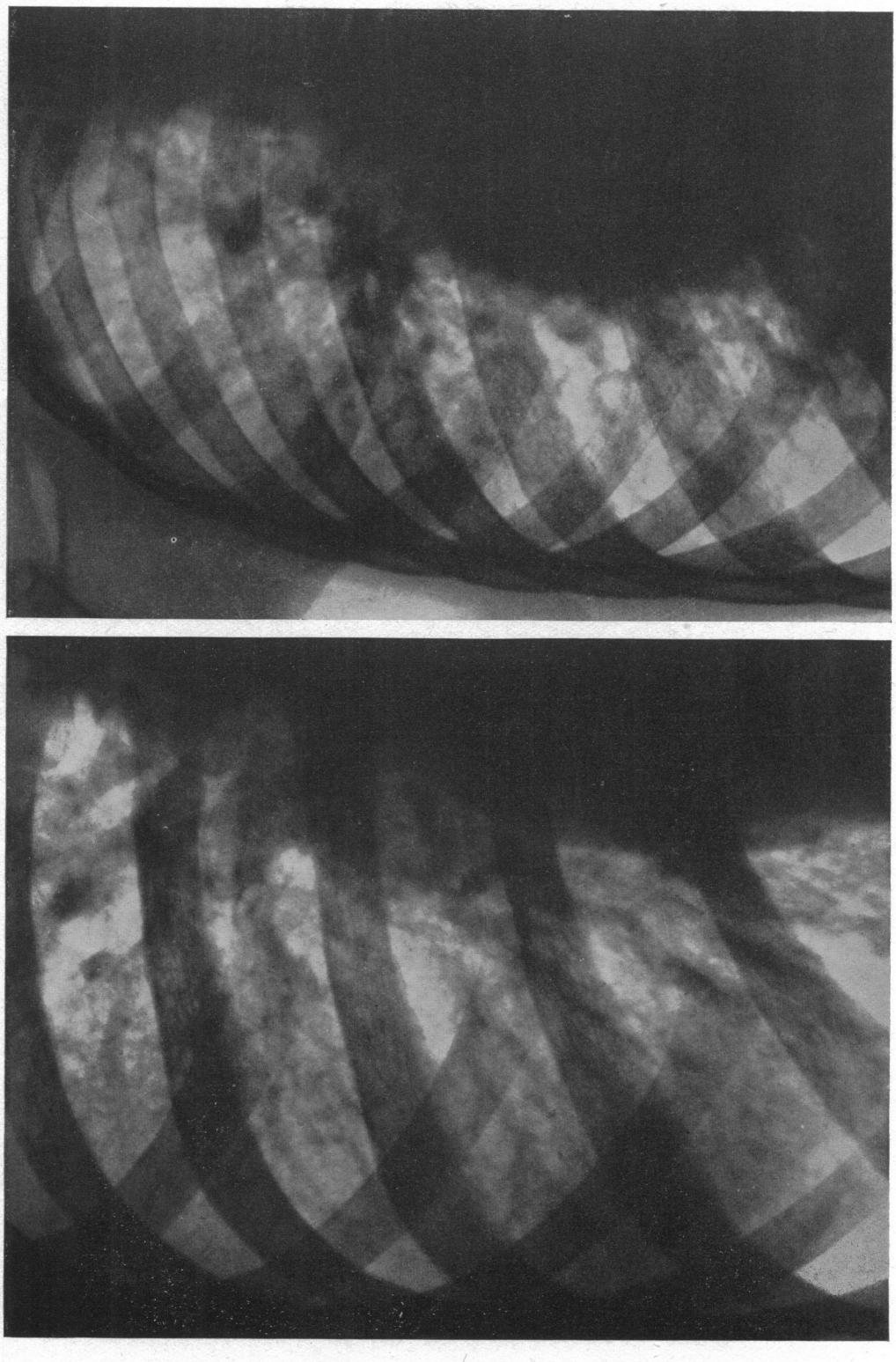


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FIG. 4.—The pleonæmic right lung fields from two patients with ventricular septal defect showing extreme mottling, as well as great visibility of the smaller branches.

(A) From a girl, aged 18, who was leading a normal life in spite of her large heart, c.t.r. 58 (m.t.d. 14·3/24·6 cm.), and pulmonary hypertension (102/61). The pulmonary branches can be seen unusually well right out to the periphery and pulsation was striking. Case 28.

(B) From a girl, aged 11, who was much disabled and has had two attacks of mild congestive failure, so that possibly some of the extra shadows here are venous. Heart greatly enlarged, c.t.r. 67 (m.t.d. 13·0/19·2 cm.), and hiding the main pulmonary arteries. Case 29.

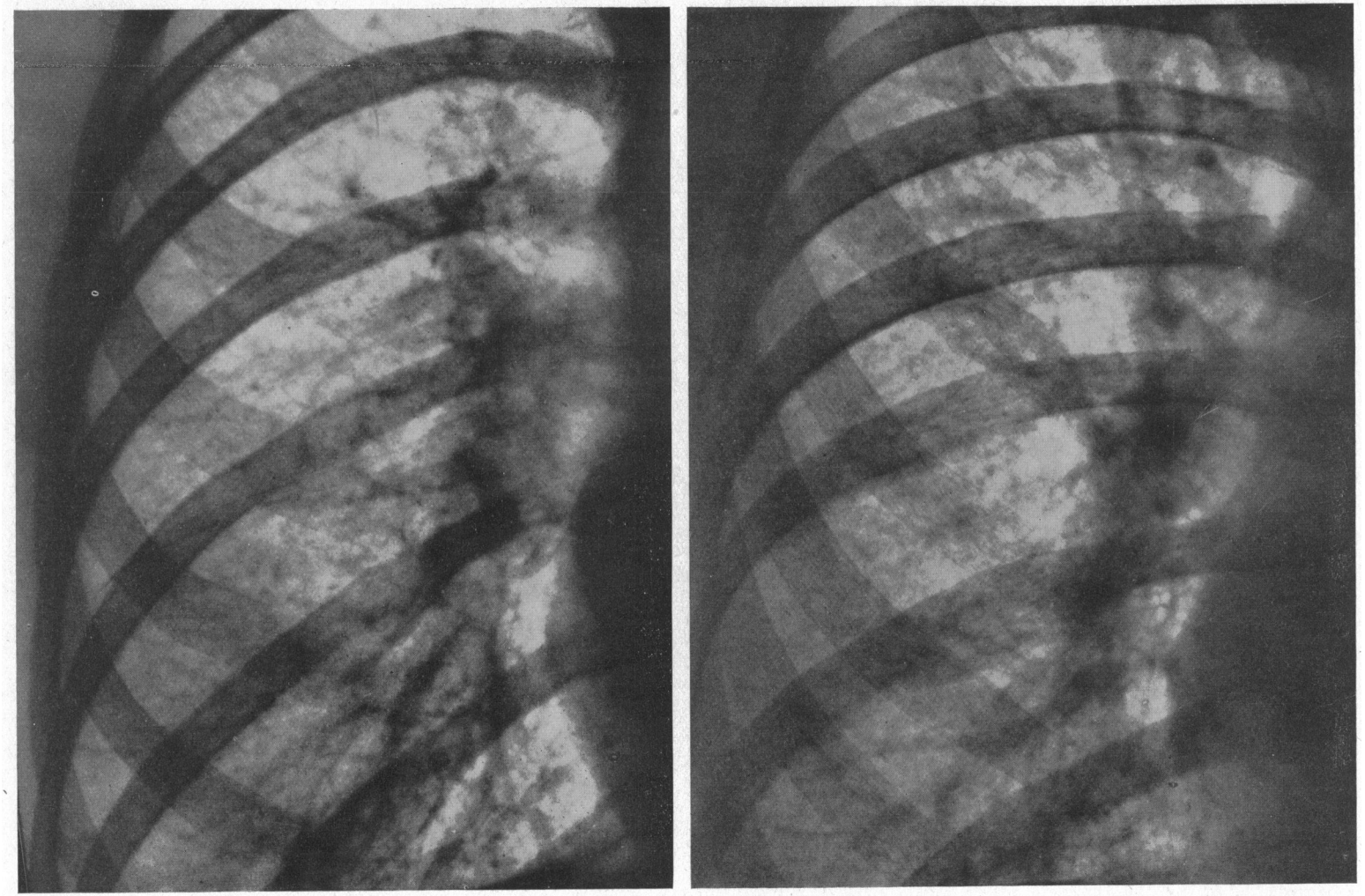

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FIG. 5.—The right lung fields from two patients with mitral stenosis, contrasting the type of shadows seen. Both these show large right pulmonary arteries and fine mottling, probably due to extravasation of blood and of heart failure cells (see text).

(A) From a woman, aged 27. The pulmonary branches are visible as well as the mottling and this was the patient with the greatest degree of visible pulsation, as it could just be seen beyond the main right P.A. C.t.r. 52 (m.t.d. 14·2/27·4). Case 54.

(B) From a woman, aged 38, who died some months later from acute pulmonary œdema. The lungs are lighter, but there is fine mottling. C.t.r. 60 (m.t.d. 16·5/27·6). Case 64.

TABLE IV

RELATIONSHIP OF SIZE AND DEGREE OF PULSATION TO BLOOD FLOW AND PRESSURE IN THE PULMONARY ARTERIES IN CASES OF MITRAL STENOSIS

Case No.	Age and Sex	Pulm. flow l./sq.m./min.	Pressure (mm. Hg)	Size of Right P.A.	Pulsation			
					Right P.A.	Middle third	Peripheral third	Hilar dance
50	F22	1·9	60	+++	—	—	—	—
51	M46	2·4	80	+++	+	—	—	—
52	F36	1·5	25	+(+)	(+)	—	—	—
53	F35	3·7	42/25	+	(+)	—	—	—
54	F27	3·9	76	++	++	+	—	—
55	F27	2·3	130/56	++	—	—	—	—
56	F27	2·1	86/41	++	—	—	—	—
57	F44	2·3	47/13	(+)+	—	—	—	—
58	F31	3·2	57/30	++	—	—	—	—
59	F30	2·3	40	++	(+)	—	—	—
60	M45	2·1	128/61	+++	(+)	—	—	—
61	M37	3·0	83/45	++	(+)	—	—	—
62	M26	1·8	75/45	+++	(+)	—	—	—
63	F34	2·5	13	+	+	—	—	—
64	F38	2·4	54	++	(+)	—	—	—
65	F39	2·8	40/22	+(+)	(+)	—	—	—
66	F26	1·7	55/15	+	—	—	—	—
67	F29	2·4	100/50	+++	(+)	—	—	—
68	F36	2·6	70/30	++	+	(+)	—	—
69	F44	3·0	18	+(+)	(+)	—	—	—

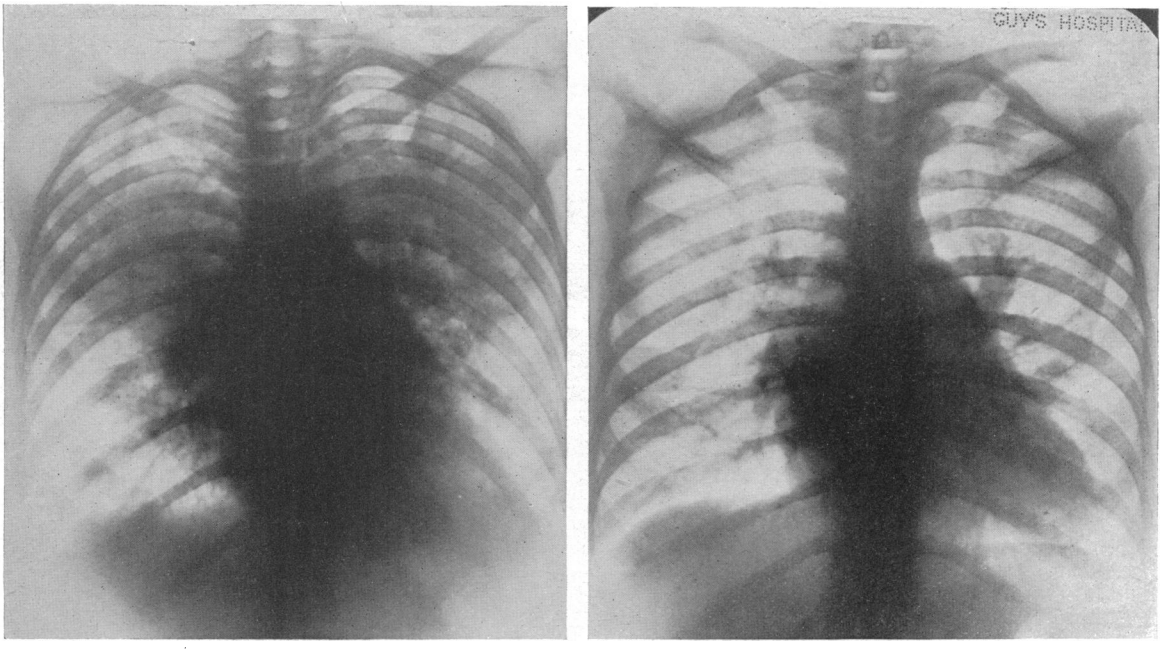
and 54 (see Table IV). There was much greater variation in the height of the pulmonary pressure, up to 130/56. There were few patients without some increase, probably because those selected for investigation had enough symptoms for mitral valvulotomy to be under consideration.

Some increase in the size of the right pulmonary artery was seen in every patient and generally it was considerable, but less than in A.S.D. Those with the higher pulmonary pressures all had large arteries but some of those with a moderate increase of pressure did not show much change. If they are divided into three groups according to whether the pulmonary pressure is greatly, moderately, or slightly increased, the height of the pressure shows some correlation with the size of the pulmonary artery, but none with the degree of visible pulsation.

In about half the cases pulsation could be seen in the right pulmonary artery, though generally it was slight and seemed no more than normal, even when the pulmonary pressure was greatly raised. Pulsation was rarely seen beyond this, but could just be seen in 3 of 20 cases in the branches in the middle third of the lung. Pulsation was never seen out to the periphery, nor any suggestion of a hilar dance. In most radiological descriptions little is said about the degree of pulsation but I think the general view is that it is absent or insignificant, though it may sometimes be seen in the pulmonary veins when there is gross mitral regurgitation.

The lung fields in mitral stenosis. The radiological features of mitral stenosis are well known and include prominence of the hilum and pulmonary arteries. Kerley (1951) describes the picture of passive hyperæmia in mitral stenosis—vessels seen end on as rounded shadows and alveoli filled with heart failure cells, the combination producing a miliary appearance like pneumoconiosis. Lendrum *et al.* (1950) have brought forward evidence that the picture often called miliary congestion is due to hæmosiderin and that this is found in groups of alveoli near the wall of the terminal bronchiole where the capillary anastomosis between the bronchial and pulmonary arteries is thought to lie.

The lung fields of two of our cases are shown in Fig. 5; in both the right pulmonary artery is increased in size. The fields look very light compared with all the previous illustrations, where the lungs were pleonæmic and the pulmonary flows were increased, although they were chosen as



A B

FIG. 6.—Lung fields contrasting the appearance of the shadows in acute pulmonary œdema, from a woman, aged 31, with mitral stenosis. C.t.r. 54 (m.t.d. 12·7/25·3). Both are portable radiograms. Case 58.

(A) During an attack of acute pulmonary œdema, showing the butterfly shadows of rather uniform density, spreading out from the hila.

(B) Three days later after her recovery. The shadow pointing to the left upper lobe is probably a vein.

examples of the mottling that is often seen in cases with orthopnœa and other pulmonary symptoms. The smaller pulmonary branches are not easily seen and the mottling is fine and does not pulsate: in fact we think it is outside the vessels and caused by extravasation of red blood corpuscles and heart failure cells. Laubry *et al.* (1948) have emphasized the association of this picture with repeated hæmoptysis, but there may have been multiple small hæmorrhages without hæmoptysis.

Although many of these patients have had attacks of pulmonary œdema, this is not the direct cause of the mottling seen. In acute pulmonary œdema the shadows have almost run together and become continuous and they spread out from the hila, generally leaving the apices and the bases clear (Fig. 6) so that they look like the wings of a butterfly.

DISCUSSION

Visible pulsation of the pulmonary arteries must, of course, represent the amount the artery is distended by the systolic output. This depends on the distensibility of the artery with a given rise of pressure (the elasticity) and the proportion of the output that has to be accommodated in that part of the artery; and this varies with the proportion of the output that can quickly pass along the artery, i.e. inversely with the peripheral resistance.

It is, therefore, unlikely that visible pulsation will be a direct measure of pulmonary flow or of pulmonary arterial pressure or even of pulse pressure. These aspects of the relationship of elasticity and resistance will be discussed by Deuchar and Knebel, but here we have tried to relate the radiological appearances to the factors of more direct clinical significance—the pulmonary pressure and the pulmonary blood flow. It seems from these considerations that the degree of visible pulsation might be greatly influenced by the amount of blood going through the lungs and that the exceptions might be explained by greater deviations from the normal as regards elasticity and peripheral resistance. Wood (1950) has calculated that the peripheral resistance is nearly always low in cases of A.S.D. but it is more variable in P.D.A. and still more so in V.S.D.

Our results are summarized in Table V. The figures for size and pulsation of the pulmonary arteries are the average number of + marks that were given to each in the group, i.e. 3 means +++ and 2 means ++, as already defined. They show that the four groups with the largest pulmonary flows have also the greatest degree of visible pulsation, except that in P.D.A. the pulsation is less than would be expected. Reynolds (personal communication) has pointed out one very important factor, the difference in the timing of the flow. In P.D.A. the flow is continuous throughout systole and diastole, though it may be more in early systole: in A.S.D. the output is sudden during ventricular systole, and there must be a more striking immediate effect which falls away again, this explaining the much greater degree of visible pulsation in A.S.D. than in P.D.A. The more frequent presence of pulmonary regurgitation may be another factor.

TABLE V

RELATIONSHIP OF AVERAGE SIZE AND DEGREE OF PULSATION TO AVERAGE BLOOD FLOW AND PRESSURE IN THE PULMONARY ARTERIES IN THE DIFFERENT GROUPS

Diagnosis and No. of cases	Pulm. flow l./sq.m./min.	Pressure (mm. Hg)	Size of Right P.A.	Pulsation			
				Right P.A.	Middle third	Peripheral third	Hilar dance
P.D.A. (10)	12·3	32/21	2·0	1·2	0·6	0	0
A.S.D. (10)	11·7	37/13	2·5	2·2	1·4	0·7	0·5
V.S.D. (10)	12·1	73/35	2·2	2·1	1·7	0·9	0·6
Eisenmenger (6)	2·5	121/64	3·0	2·0	1·0	0·3	0·3
Pulmonary hypertension (4)	2·3	109/62	2·4	1·2	0·6	0	0
Transposition (6)	6·2	87/50	2·9	2·0	1·5	0·9	0·5
Mitral stenosis (20)	2·5	76/37	2·1	0·5	0·1	0	0

They also show that increased pulsation and some increase in size of the pulmonary artery can take place without any significant rise of pulmonary pressure.

The cases of mitral stenosis and, to a lesser extent, those of pulmonary hypertension show that a large rise of pulmonary pressure does little or nothing to produce visible pulsation. Comparing the degree of pulsation in A.S.D. and V.S.D. also shows that when there is a large pulmonary flow an increase of pressure causes little further increase in visible pulsation. On the other hand, in Eisenmenger's complex there is generally no increased pulmonary flow to account for the visible pulsation so that this forms an exception.

Table V also shows that the pulmonary arteries are largest in Eisenmenger's complex and in transposition, followed by atrial septal defect and by pulmonary hypertension, so that enlargement of the pulmonary artery is caused most often by increased pressure but may be caused by an increased flow.

Healy, Dow, Sosmann, and Dexter (1949) have studied this in a similar way and have helped to elucidate the relationship of pleonæmic lungs to increased pulmonary blood flow and pressure. They classified the size of the pulmonary artery and its branches into four grades, and the degree of pulsation in these vessels into two grades.

A.S.D. and P.D.A. provided the main examples of increased pulmonary blood flow. In 7 cases of A.S.D. the blood flows were increased, generally ranging from 7 to 15 litres/sq.m./min. and the pressures were not much raised (see Table VI). The pulmonary artery was generally dilated, grade 3 or 2, and the pulsation was generally grade 2. The size of the branches varied from grade 3 to the upper limits of normal, and the pulsation of the branches varied from grade 2 to doubtful. In 9 cases of P.D.A. the changes were similar but much less. The pulmonary flows were less increased, generally ranging from 3 to 7 litres/sq.m./min., but the pressures were about the same. The pulmonary arteries were less dilated, from grade 1 to normal, and the pulsation

was also about grade 1. In their Table I there was good individual correlation between the observed changes and the increase of pulmonary flow. There seemed to be a critical level so that if the flow was above 7 litres/sq.m./min., large pulmonary arteries and increased pulsation were seen, but below this they were unlikely to be seen.

As examples of a raised pressure without increase of pulmonary flow they used cases of Eisenmenger's complex and of mitral stenosis. In Eisenmenger's complex, the blood flows varied from 2 to 4 litres/sq.m./min. and the pressures were often as high as 130/50. In mitral stenosis the flows varied from 2 to 4 litres/sq.m./min. and the pressures were more variable but often much increased, from 40/20 to 115/60. In both these groups, changes in the size of the arteries were seen and in Eisenmenger's complex increased pulsation of about the same degree as in P.D.A. but much less than in A.S.D. When both the pressure and the flow were increased (some cases of A.S.D. and P.D.A.) they found the degree of enlargement and of pulsation both increased, even more than in these conditions separately.

They concluded: "We are unable to distinguish the effects of increased flow with normal pressure from increased pressure with normal flow," but also stated that hyperactivity (increased pulsation) was not quite such a striking feature of increased pressure as of increased flow, but might occur and might be quite marked. My examination of their figures, however, suggests that increase of pulsation is not nearly such a striking feature of increased pressure as it is of increased flow. In Table VI I have summarized their results, marking numerically the degrees into which they grade pulsation and the size of the pulmonary artery and giving an average figure. In the first two

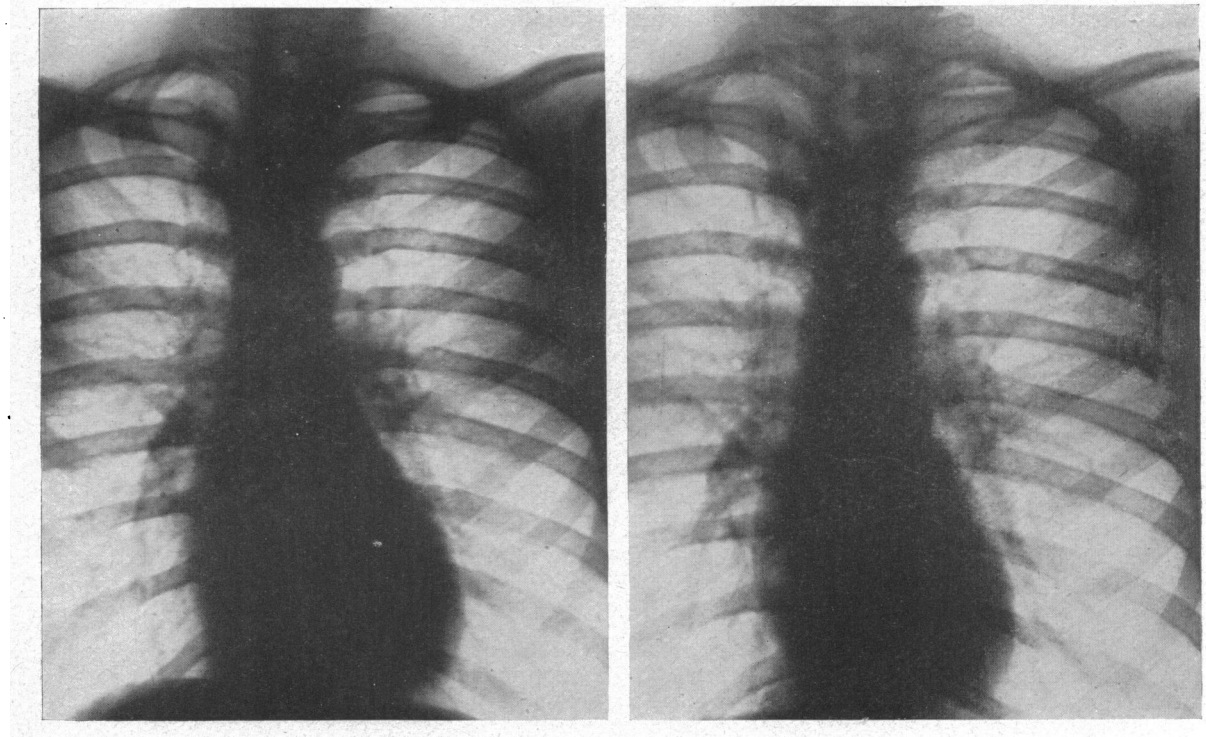
TABLE VI

THE RELATIONSHIP OF PULMONARY ARTERY SIZE AND PULSATION TO PULMONARY FLOWS AND PRESSURES MODIFIED FROM HEALEY, DOW, SOSMANN, and DEXTER (1950)

Diagnosis	No. of cases	Size of Pulm. artery		Degree of Pulsation		Pulm. flow l./sq.m./min. (Usual range, and average in brackets)	Pulm. artery pressures (Usual range)
		Right P.A.	Branches	Right P.A.	Branches		
		Increased flow without increased pressure					
A.S.D.	7	2·3	1·6	1·6	1	7–15 (10·3)	35/15 to 20/7
P.D.A.	9	1·1	0·9	0·9	0·3	3–7 (4·7)	35/12 to 20/10
		Increased pressure without increased flow					
Eisenmeng.	8	2·5	2·0	0·9	0·5	2–4 (2·9)	130/65 to 100/55
Mitral Stenosis	8	2·0	2·0	0·2	0	2–4 (3·1)	115/60 to 40/20
		Increased flow and pressure					
A.S.D.	5	2·6	2·8	1·5	1·4	7–19 (12·5)	85/40 to 45/20
P.D.A.	4	1·5	2·0	0·4	0·4	9–20 (12·5)	60/40 to 45/25

groups where the pressures were the same but the flows greater, A.S.D. showed larger vessels and more pulsation than P.D.A. This was also true when the pressures and flows were both greatly increased. The Eisenmenger cases showed much more pulsation than the mitral stenosis cases although the flows were the same. This might be an intrinsic difference or might be because the pressures were higher; the former seems more likely because separating the A.S.D. cases into those with high and those with normal pressures did not make much difference to the degrees of pulsation.

It is interesting that these figures are so similar to mine because many of my patients were children and none of those of Healey *et al.* were under 9 years of age. Another reason for analyzing their



A B

FIG. 7.—Teleradiograms from a healthy student (A) before and (B) immediately after violent exercise, showing that in normal conditions the pulmonary flow can be greatly increased without producing the picture of pleonæmic lungs.

figures was lest my subjective judgement of size and pulsation had been increasingly biassed by the views I had formed.

The increase of pulmonary flow with exercise. If increased pulmonary flow causes the visible pulsation in the pulmonary arteries, the normal increase of pulmonary flow after exercise might demonstrate these changes. Some observations carried out to test this showed that normally the pulmonary flow can be greatly increased without any change, so that it can only be increased flow maintained over a long period that is responsible.

Two healthy young men who were in moderate training ran up the three flights of stairs above the X-ray room five times in quick succession, and a film was taken just before and immediately after. As this exercise meant they ran up and down 455 stairs in rather over four minutes, there must have been a large pulmonary flow, probably four times its normal. As further evidence of this, the pulse rate of the first student was still 134 a minute after the completion of the second X-ray, and that of the second, which had been 70 before exercise, fell from 170 to 124 in the three minutes after.

In spite of this considerable increase of the pulmonary flow, no difference could be seen in the films taken before and afterwards: apart from the marking, the films taken after exercise could only be distinguished by the greater prominence of a small shadow, probably the inferior vena cava. The second student repeated this exercise on another day so that he could be screened immediately after it. There was no obvious difference, though possibly it was just a little less difficult to see the normal pulsation in the main right and left pulmonary arteries.

SUMMARY

The size of the right pulmonary artery and the degree of visible pulsation in this vessel and in the smaller branches of the middle and peripheral thirds of the lung fields have been classified in four grades in various types of congenital heart disease and in mitral stenosis. They have been compared with the pulmonary blood flow and pulmonary arterial pressure as determined by cardiac catheterization. Visible pulsation is in general evidence of increased pulmonary flow.

Pulsation in the right pulmonary artery is greatest in atrial and ventricular septal defect, in Eisenmenger's complex, and in transposition of the aorta and pulmonary artery: it is much less in patent ductus arteriosus and in pulmonary hypertension, and insignificant in mitral stenosis. Pulsation in the peripheral third of the lung and a hilar dance are only seen in atrial and ventricular septal defects and in transposition, and occasionally in Eisenmenger's complex.

The main pulmonary arteries are largest in cases of Eisenmenger's complex and transposition. They are dilated but not quite so much in atrial septal defect, in pulmonary hypertension, in ventricular septal defect, in mitral stenosis, and in patent ductus, in this order. The range of variation in these groups is much the same, except that all the cases of patent ductus fall in the lower part of the range.

The size of the pulmonary arteries is influenced both by increased pressure and by increased flow, and still more when both are increased. Visible pulsation is seen when there is an increased flow, whether the pressure is increased or not. In septal defects, increase of pressure has no further effect on pulsation but it may in some other conditions, e.g. Eisenmenger's complex.

A.S.D. and V.S.D. have more effect on size and much more effect on pulsation than P.D.A. has, independently of the increase of flows and pressures, because a large part of the cardiac output is forced into the arteries suddenly during systole instead of more gradually during systole and diastole. Eisenmenger's complex has more effect on pulsation than pulmonary hypertension or mitral stenosis has, independently of flows and pressures, possibly because an increased flow in the past has led to this change.

A large pulmonary blood flow increases the size of the pulmonary artery and the degree of visible pulsation, whether the pulmonary arterial pressure is increased or not. A high pulmonary pressure, without a large flow, increases the size of the right and left pulmonary arteries, but has little or no effect on the degree of visible pulsation, except in Eisenmenger's complex.

I am glad of the opportunity of thanking Sir John Parkinson for all I have learnt from him about the radiology of the heart. I am also grateful to Mr. E. L. York, classical tutor of New College, Oxford for suggesting the word oligæmic (ὀλιγαιμος) and to Dr. H. St. H. Vertue for adding the contrasting pleonæmic (πλεοναιμος).

I should also like to thank Dr. Deuchar for the results of cardiac catheterization in most of the cases; Drs. Holling and Zak in Cases 10, 14, 17, 19, 32, 41, 42, 45 and most of the cases of mitral stenosis; and Dr. Paul Wood in Cases 16, 29, 36, 37, 54, and 59.

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